Case history

Evelyn Dunn has been under your dental hygiene care for the past 5 years. Because of the patient's good oral hygiene, she was placed on a 12-month recall. Evelyn is now 19 years of age and has presented for her yearly dental examination and cleaning after returning from travelling for 18 months after completing year 12 of High School.

IS YOUR KNOWLEDGE UP-TO-DATE?

Social history

The patient smokes three cigarettes a day because her last birthday. She drinks alcohol on Friday and Saturday nights with her friends.

Medical history

Evelyn has a negative medical history. She takes a daily over-the-counter multi-vitamin and protein shakes twice a day as a meal substitute as she is concerned about her weight.

Extra-oral examination

No significant findings all structures within normal limits.

Intra-oral examination

At the patient's last dental visit, the molar and anterior incisor teeth exhibited localized periodontal pocketing of 4 mm with no signs of gingival inflammation.

Periodontal probing measurements are 7 mm localized to the interproximal sites of the incisor-molar sextants, 7–10 mm facial maxillary incisors and clinical attachment loss over 3 mm with Class II mobility in the cuspids, bicuspids and maxillary molars.

Radiographic image

Panoramic and vertical bite-wings radiographs revealed an overall pattern of moderate-to-severe horizontal bone loss with localized catering. The maxillary incisors and the mandibular molars exhibited widened periodontal ligament spaces.

Questions

1 The rapid destruction of the periodontium is indicative of which classification of periodontal disease?

2 Discuss the diagnostic characteristics and therapeutic management of the patient's oral health status.

Rationale

1. The rapid destruction of the periodontium is indicative of which classification of periodontal disease

Children and adolescents are subject to several periodontal diseases. Although there is a much lower prevalence of destructive periodontal diseases in children than in adults, children can develop severe forms of periodontitis (1). Epidemiological surveys in young individuals have been performed in many parts of the world and among individuals with a widely varied background (2). For the most part, these surveys indicated that loss of periodontal attachment and supporting bone is relatively uncommon in the young, but that the incidence increases in adolescents aged 12-17 years when compared with children aged 5-11 years (3). The worldwide infection rate of aggressive periodontitis among children stands at 2% according to World Health Organization (WHO) statistics (4). The prevalence of early oral microbial infection around the world is growing and can be considered to be at epidemic proportions. This epidemic strikes at the heart of many societies and is arresting children's social development (5). The clinical manifestation of the disease can result in the protrusion of maxillary incisors following periodontal attachment loss, extensive destruction of anterior papilla, and loss of maxillary central incisors are possible effects of early oral microbial infection. They all cause serious damage to the facial expression, which can have a significant psychological and emotional impact on young patients. Early oral microbial infection damages the self-esteem, confidence, and well-being of children and young people (5).

The term 'early-onset periodontitis' (EOP) was used in the 1989 American Academy of Periodontology (AAP) and 1993 European classification as a collective designation for a group of dissimilar destructive periodontal diseases that affected young patients (6). Additional terms that have been used to describe this diverse group of clinical entities include localized and generalized forms of prepubertal periodontitis (PP), localized and generalized forms of juvenile periodontitis (LJP and GJP, respectively), and rapidly progressive periodontitis (RPP) (7). Recently, a proposal for a new classification of periodontal diseases has been published, which recommends that EOP be reclassified as aggressive periodontitis and that its current subclassification into PP, JP and RPP be discarded (8). Classification systems are necessary to provide a framework in which to scientifically study the aetiology, pathogenesis and treatment of diseases in an orderly fashion. A classification based on infection as the principal aetiology of periodontal diseases divides categories based on gingival inflammation, periodontal attachment loss and recognizes health, gingivitis, and periodontitis as separate entities (6). Children and adolescents can have

any of the several forms or periodontitis as described in the proceedings of the 1999 International Workshop for a Classification of Periodontal Diseases and Conditions (aggressive periodontitis, chronic periodontitis, and periodontitis as a manifestation of systemic diseases (2).

The 1999 International Workshop classification system includes a greater variety of disease categories that base the diagnosis on clinical history and findings, relying less on age of onset as a major criterion (9).

Classification of periodontitis

- 1 Gingival Diseases
- 2 Chronic periodontitis
- 3 Aggressive periodontitis
- 4 Periodontitis as a manifestation of systemic diseases
- 5 Necrotizing periodontal diseases
- 6 Abscesses of the periodontium
- 7 Periodontitis associated with endodontic lesions

8 Developmental or acquired deformities and conditions The age of onset for PP is before puberty. For JP, it corre-

sponds to the circumpubertal years and for RPP, it ranges from 20 to 35 years of age. In contrast to adult periodontitis (AP), the ages on onset for different forms of EOP vary, but all occur prior to age 35 years (10). The dominant periodontitis paradigm holds that aggressive periodontitis (AgP) 'is a specific type' of periodontitis with clearly identifiable clinical and laboratory findings, which make it sufficiently different from Chronic Periodontitis encompasses distinct types of periodontitis that affect people who, in most cases, otherwise appear healthy. It tends to have a familial aggregation and there is a rapid rate of disease progression. Aggressive periodontitis occurs in localized and generalized forms (11). AgP is believed to occur mainly, although not exclusively (8), in adolescents and young adults (12).

Localized aggressive periodontitis (LAgP) patients have interproximal attachment loss on at least two permanent first molars and incisors, with attachment loss on more than two teeth other than first molars and incisors. Alveolar bone destruction proceeds more rapidly than in the localized form. In the localized form, particular teeth are affected and alveolar bone loss can progress faster than in chronic periodontitis (13).

Usually, generalized aggressive periodontitis starts at the point of tooth eruption (13). Patients with generalized aggressive periodontitis (GAgP) exhibit generalized interproximal attachment loss including at least three teeth that are not first molars and incisors. Severity can be characterized on the basis of the amount of clinical attachment loss (CAL) as follows: slight = 1 or 2 mm CAL, moderate = 3 or 4 mm CAL, and severe \geq 5 mm CAL. At least two observations separated in time for the assessment of the rate of periodontal destruction and, hence, a diagnosis of AgP, it is commonly accepted practice to use an assessment of disease-for-age as a surrogate measure of the progression rate (6). As early diagnosis ensures the greatest chance of successful treatment, (14) it is important



Fig. 1. Clinical signs of gingival health with periodontal pocketing.

that children receive a periodontal examination as part of their routine dental visits.

Several bacterial species residing in a biofilm on tooth surfaces referred to as dental plaque have been closely associated with periodontitis (15). Lack of clinical inflammation; affected tissue may have a normal clinical appearance but probing will reveal deep periodontal pockets on proximal surface(s) of affected teeth (Fig. 1). Relatively spare bacterial plaque; disease severity seems exaggerated given the relatively light amount of bacterial plaque (16). However, different forms of periodontal disease may have distinct microflora colonizing the periodontal pockets (17), which require a series of clinical examination and diagnostic procedure to determine the appropriate treatment and management of the disease.

Periodontal diseases are among the most frequent diseases affecting children and adolescents. Dental clinicians must be aware of the prevalence, diagnostic characteristics, microbiology, host-related factors, and therapeutic management of each of these disease entities. It is well known that the primary aetiology of periodontal diseases is bacterial plaque. It is important that when managing periodontal diseases in young individuals, the dentist should rule out systemic diseases than can affect host defence mechanisms (18).

2. Discuss the diagnostic characteristics and therapeutic management of the patient's oral health status

Periodontitis is currently diagnosed almost entirely on the basis of its clinical manifestations: signs of gingival inflammation (e.g. redness and swelling), periodontal pocket depth and level of periodontal attachment or amount of alveolar bone loss. However, alveolar bone and periodontal attachment loss cannot be used to predict susceptibility to future disease progression or dictate appropriate treatment plans (19).

As the aetiology of and risk factors for periodontal diseases are multifaceted, the most important steps for patient evaluation include a comprehensive clinical and radiographic analysis (20). But when examined by eye, radiographs can only reveal changes in bone after 30–50% of the mineral has been lost (21). In addition, radiographs cannot be taken at each visit because of the radiation exposure to the patient. Most importantly, radiographic examination and clinical probing can only provide information about the level of tissue destruction and disease severity. Therefore, conventional radiography is not useful for evaluating disease activity or the risk of disease progression (19). Compared with traditional radiography (DSR) are more sensitive to small bony changes in the alveolar crest with a higher degree of diagnostic accuracy (19, 22). However, DSR is not used much in clinical practice because it is a difficult technique to master, but is used frequently in clinical research (20).

In fact, alveolar bone and periodontal attachment loss represent only the results of the destructive aspects of the host defence mechanisms responding to opportunistic infections by bacteria present in the gingival sulcus, as well as the direct effect of virulence factors of periodontal pathogens (19). In the diagnosis of aggressive periodontal disease, identification of factors including cigarette smoking, modifying systemic risk like diabetes mellitus (20), immune defects and the microbial flora, control of the diseases may not be possible in all instances. In some cases, blood test is conduced prior to treatment to exclude systemic conditions such as hypophosphatasia, neutropaenia and leukaemia (13). Diagnostic and prognostic tests to address these problems have been extensively sought. Tests are needed to define active versus inactive sites and to identify sites within periodontitis patients who may require additional/alternative treatments (19). The suggested method incorporates a multidisciplinary approach incorporating clinical laboratory evaluation with conventional concepts of periodontal

pathogenesis and therapeutics to diagnose and effectively treat early-onset periodontitis (currently classified as Aggressive Periodontitis) (7).

Adjunctive tests for the evaluation of periodontal diseases are divided into three main groups: gingival crevicular fluid assays, microbial tests and genetic assays. A summary of the adjunctive tests is provided in Table 1 (20).

Therefore, these new assessment methods shift the focus from determining disease severity to evaluating disease progression of the risk of future periodontal breakdown, basically, quantifying ongoing disease activity. Currently, however, few of these tests are available commercially, and no test is available that will definitely determine if a site is actively losing attachment and bone or will lose attachment in the future (20). There are no specific guidelines to follow, but some factors to consider include cost of testing, age of the patient, patient motivation and systemic health of the patient as the host response contributes to the development and progression of the disease. Therefore, no microbial or host response test will definitely determine the initiation or progression of disease (20).

DNA probe analysis can be helpful in these patients, providing rapid results within 24 h. Assessment of the genetic susceptibility of a patient or of children or parents with advanced periodontal disease my soon prove to be an important tool in the initiation of preventive measures (20). However, subgingival plaque samples from affected teeth can be assessed to determine the associations between the presence and quantities of a number of putative periodontopathogens and the clinical periodontal conditions (12). The microbial test shows the presence of elevated proportions of non-motile, facultatively

Table 1. Adjunctive tests

Advantages and limitations of selective adjunctive tests		
Test	Advantages	Limitations
Culture	Identifies specific microorganisms Only test available that determines antibiotic sensitivity	Expensive and time-consuming Technique sensitive; diverse data from different labs (use of different media, sampling, transport) Cell vitality must be preserved during transportation to lab
DNA probe assays	Highly sensitive and specific for targeted periodontal pathogens Viable organism not needed	Need special disposable procedures for radioactive waste Does not determine antibiotic sensitivity of the bacteria being tested
	Samples sent by mail after collection chairside Rapid identification (18 h)	Certain bacterial species may not be detected because of highly sensitive nature of the test Performed in a lab
Microscopic test (phase-contrast microscopy)	Good for chairside patient awareness of the importance of bacteria in periodontal disease Motivational tool for patient's plaque-control habits	Does not specifically identify bacterial species, only shape, size, and mobility Not suitable to monitor disease activity
Gingival crevicular fluid (GCF) assays	Rapid and inexpensive	Cannot identify specific bacteria, but allows for rapid assessment of bacterial enzymatic activity
	Technique insensitive, so easy to perform chairside Good for screening purpose In-office use	Need sufficient amount enzymes

anaerobic, Gram-negative rods, which are known periodontal pathogens including *Actinobacillus actinomycetemcomitans*, *Prevotella intermedia* and *Porphyromonas gingivalis* (7, 23).

Serum antibody analyses of blood samples obtained by venipuncture can be conducted to test serum levels IgG, IgA and IgM against 6 periodontal bacteria (24). The most severe forms of aggressive periodontitis showed significant elevations in both *IgG* and *IgA* antibody titres to *P. gingivalis* and *A. Actinomycetemcomitans* and *IgA* antibody titres to *P. intermedia* compared with healthy control in research investigations. In some cases, rather than being protective, high antibody titres may be associated with increased disease severity (24, 25). In addition, elevated leucocyte counts are related to the patient's severe periodontal infection (7). In histological examination, lymphocytes and plasma cells had infiltrated into the lamina propria mucous of the gingiva, and neutrophils in the gingival connective tissue, which are features similarly found in typical adult chronic periodontitis (13).

When treating younger periodontitis patients, it is extremely important to educate the patient and his or her parent/guardian concerning the potential consequences of non-compliance (7), as current modalities for managing periodontal disease of children and adolescents may include antibiotic therapy in combination with non-surgical and/or surgical therapy (14) with the primary goal of the treatment to control the periodontal infection, reduce probing depths and gain clinical attachment (26). The treatment methods for AgP may be similar to the treatment recommendation used for chronic periodontitis. These methods should include oral hygiene instruction and reinforcement and evaluation of the patient's plaque-control. Appointments of scaling and root planning by quadrants are required to remove microbial plaque and calculus; control of other local factors, occlusal therapy as necessary and periodontal maintenance (11). The literature indicates that mechanical debridement alone is only moderately effective in suppressing periodontal pathogens in general and ineffective in eliminating A. actinomycetemcomitans (26).

Alternate antibiotics may be required, based upon the character of the pathogenic flora, in GAgP patients who have failed to respond to standard periodontal therapy, laboratory tests of plaque samples may identify periodontal pathogens that are resistant to antibiotics typically used to treat periodontitis. It has been suggested that follow-up tests after additional antibiotic or other therapy is provided may be helpful in confirming elimination of targeted pathogenic organisms (27). Based on the results of antibiotic sensitivity assays performed at the time of the microbiological analysis, patients are placed on metronidazole (250 mg t.i.d) in combination with amoxicillin (500 mg q.i.d) for 8 days. This regimen has been shown to be 97% effective in the elimination of A. actinomycetemcomitans (28). The local delivery of antimicrobials, and other systemic antibiotics such as tetracycline, doxycycline, metronidazone, combination of metronidazole and amoxicillin, or combination of metronidazole and Augmentin (amoxicillin + clavulanic acid) (18) have also provided successful treatment outcomes. However, in very young patients, the use of tetracyclines may be contraindicated because of the possibility of staining of teeth. Alternative antimicrobial agents or delivery systems may be considered (11). The management of AgP requires re-evaluation appointments scheduled monthly. At each appointment, supragingival scaling is repeated and probing depths and attachments levels measured and when indicated, antimicrobial irrigation of the periodontal pockets. Re-instruction and remotivation in effective oral hygiene is conducted. Eight months after the initiation of treatment, plaque samples are taken for microbiological re-evaluation (7).

Young individuals generally have excellent healing potential. Therefore, in JP (currently classified as Aggressive Periodontitis) patients, the combination of systemic antibiotics and regenerative surgery is often successful in treating infrabony defects and early furcation involvement (18). Successful treatment of aggressive periodontitis depends on early diagnosis, directing therapy against the infecting microorganisms and providing an environment for healing that is free of infection (14), thereby arresting the progression of disease and preserving the dentition in comfort, function and appropriate aesthetics and to prevent the recurrence of disease (11).

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Photograph courtesy of Associate Professor Ivan Darby, Head, Periodontics, Melbourne Dental School, Faculty of Medicine, Dentistry and Health Sciences University of Melbourne, Australia.

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